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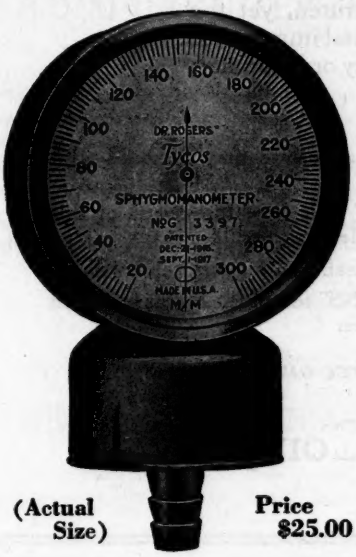

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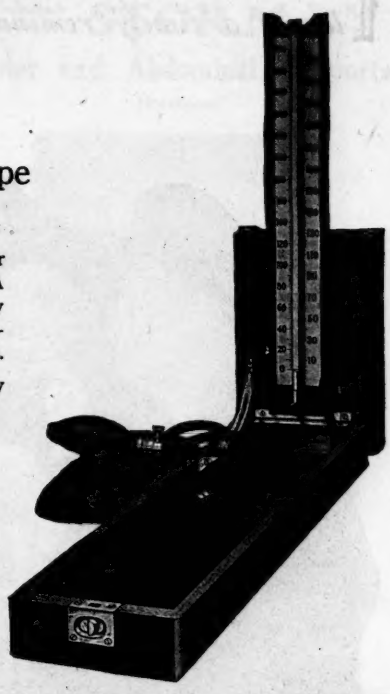

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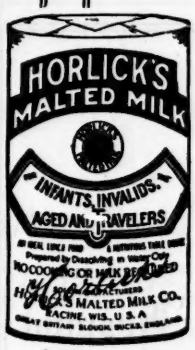

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ORIGINAL ARTICLES

A STUDY IN EXPERIMENTAL ANIMALS OF THE CAUSE AND TREATMENT OF THE SERIOUS REACTIONS FOLLOWING QUINIDINE.*

By Burgess Gordon, M.D.

Philadelphia, Pa.

During the past year in the Medical Clinic of the Peter Bent Brigham Hospital a number of studies have been made on the cause and treatment of serious reactions following the administration of quinidine sulphate. Interest in this investigation was aroused because of three fatalities that occurred in the wards of the clinic during quinidine therapy. Post mortem examination of these patients failed to demonstrate the cause of sudden death and in one case where death was not instantaneous there was no explanation of the curious toxic state, the appearance of shock and the unusual respiratory distress existing for some hours before the fatal termination.

A review of the literature indicates considerable confusion as to whether the heart or respiratory system is primarily affected. Embolic phenomena,[1] increased irritability of the ventricles,[2] intoxication of the respiratory centre,[3] failure of the circulation,[4] and other causes,[5] have been given as explanations for sudden or unexpected deaths following quinidine administration.

It was with the purpose of studying the mechanism of death and thereby obtaining information as to whether there was any means of preventing such catastrophies that the following investigation was made. Adult male cats were used in the experiments. On account of the technique and other details of the study may be found in the original paper.[6]

*From the Medical Clinic of the Peter Bent Brigham Hospital and the Department of Medicine of the Harvard Medical School.

*Read before the Rhode Island Medical Society, Providence, R. I., June 4, 1925.

The first experiments were carried out on animals of known weight to determine the lethal dose of the drug. They indicated, first of all, that there was some relation between the weight of the animal and the size of the dose. It was found that a single injection of between 25 and 30 milligrams of quinidine per kilogram was fatal. The total minimal lethal dose, however, was 45 milligrams per kilogram if 15 milligrams were given every six minutes. It was possible, by giving still smaller doses over a period of two hours, to administer 100 milligrams per kilogram before the lethal effect was obtained. This total dose was four times as great as the minimal lethal amount when one single dose was given. In the non-lethal experiments a single injection of 20 milligrams per kilogram was found to cause no appreciable change in the respiration except occasionally a moderate slowing. This was considered within the margin of safety for administration.

The blood pressure observations were striking in that a sudden drop of the pressure occurred immediately after the first injection of quinidine. This degree of fall varied between forty and eighty millimeters of mercury, and was followed quickly by a gradual although incomplete return to the normal level. There was a tendency for the blood pressure to remain low for a longer time when the dose was large, the return also being less complete. The fall in pressure which was so constant was probably due to two factors, the most important role being played by a peripheral vasomotor depression and the other by the toxic effect on the heart itself.

In studying the direct action of quinidine on the heart, numerous electrocardiograms and roentgenograms were taken at different times following administration. In general, it was found that small doses of quinidine caused transient changes in the ventricular complexes. These changes disappeared after the first injection of quinidine, but became progressively more marked with repeated injections and with a gradually diminishing degree of recovery. The final tracings frequently showed

bizarre ventricular effects. Successive roentgenograms taken after one small injection of quinidine showed a diminution in the size of the heart with a gradual return to normal. If the dose was larger or about 22 milligrams per kilogram of body weight, there was also a contraction immediately after administration, but this was followed by a sudden marked dilatation, then a gradual return to normal. This sudden dilatation was thought to be the result of a decreased elasticity of the heart muscle caused by the direct action of quinidine on the heart,[7] and the contraction was due to the action of quinidine on the peripheral vessels, producing a fall in pressure.[8]

I have already stated that 20 milligrams of quinidine per kilogram of body weight was found to cause no appreciable change in the respiration except occasionally a moderate slowing. On the other hand, when a single dose of 25 milligrams per kilogram was given, which was in the vicinity of the lethal dose, there was frequently a brief cessation of the respiration followed by a gradual return to normal. In giving larger doses of quinidine, it was found to be an invariable experience for the respirations to cease and for the heart to continue beating for some time after the respiration had stopped.

Throughout the experiments the asthenic appearance of the animals closely resembled the picture of intoxication of the one patient who died at the P. B. B. H. following the oral administration of small doses of quinidine during the last few hours of life. In the animals there was a curious livid appearance of the lips and from time to time they tossed their heads from side to side and threw their legs about limply. In was not uncommon for the cats to develop short periods of convulsions, and in some there was relaxation of the sphincters.

In the first experiments it was the impression that the cats were dying a cardiac or circulatory death. Such drugs as oubaine, strophanthin and digitalis were given intravenously when symptoms of catastrophe appeared. The drugs were also used before the injection of quinidine with an idea of preventing the deleterious effect on the heart and circulation. The harmful effect of quinidine was neither prevented nor removed by the use of these drugs. Suspecting that the vital centers failed to receive a sufficient blood supply because of the low blood pressure, a number of the animals were placed head down when the breathing stopped, but there was no improvement in the respirations. It was thought that caffeine might be of value if the mechanism of the quinidine phenomenon were a respiratory paralysis, as caffeine is felt to have a stimulating effect on the respiration.[9] Thus a group of animals were given a moderately large dose of quinidine, a dose sufficient to produce respiratory embarrassment. At the point where the cat was breathing poorly an injection of caffeine sodium benzoate was given (about 5 milligrams per kilogram). In most instances the normal breathing returned. In other experiments the cats received sufficiently large doses of quinidine to produce complete respiratory failure. One minute after breathing had stopped an intravenous injection of caffeine was made. This was followed in about one-half of the series by a return of normal breathing. A definite beneficial effect of caffeine on the untoward depression of the respiratory mechanism following quinidine was shown in one animal which developed failure of the respiration after 25 milligrams per kilogram. On the next day this animal was given caffeine, which was followed in ten minutes by the injection of 30 milligrams of quinidine per kilogram of body weight. There was practically no change in the respirations except a moderate slowing. This showed that caffeine prevented the development of respiratory failure if given before quinidine administration.

Not all cats responded as satisfactorily to caffeine, so other methods of resuscitation were studied. Artificial respiration by means of chest massage was quite successful if the animals were not hopelessly intoxicated by quinidine and when the respiratory movements were made in a slow and careful manner. A most dependable method was found in intratracheal artificial respiration. Cats given fatal amounts of quinidine, either in a single massive dose or in small repeated doses which were sufficient to produce cessation of the respiration for as long as two minutes, were saved by this procedure. Some cats, in which automatic breathing had stopped, were given artificial respiration for from fifteen to twenty minutes before normal breathing returned. A combination of

caffeine and artificial respiration was found to be not only dependable but hastened recovery in most instances.

A a result of these experiments, it was found that quinidine has a peripheral depressant effect on the respiratory apparatus of cats which proves fatal at a time when the heart, though somewhat intoxicated, is still viable. Stimulation of the respiratory mechanism by caffeine, combined with the use of artificial breathing, provides a satisfactory means of preventing such fatalities. The study, although not attempting to explain the theory of exitus caused by embolic phenomena, throws light on one type of death during quinidine administration.

SUMMARY

The above experimental data accounts for some of the conflicting views expressed by previous observers on the cause of death during quinidine administration. There is positive evidence of heart muscle intoxication and of a vaso-dilator effect following the use of the drug. However, the respiratory paralysis cannot be explained in any other way than as a specific effect on the respiration and not as the indirect result of the low blood pressure. Successful methods of resuscitation substantiate this view. Experience in the clinic with one patient, who died following quinidine therapy and who showed respiratory embarrassment some hours before death, makes it seem likely that means of resuscitation used in these experiments might be applicable to human cases. It is suggested that artificial respiration be employed with caffeine intravenously in doses comparable to the amount used in the animal experiments (about 0.5 grams intravenously for the average adult).

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"A REPORT ON FIFTY CASES OF PYELITIS IN CHILDREN"*

BY DR. ROBERT M. LORD

PROVIDENCE, R. I.

As I must necessarily turn somewhat to the current literature for experimental work on pyelitis, I wish to give a short summary of a few special phases of the problem of pyelitis which have been worked out by various research men, and then turn to a consideration of the fifty cases which I summarized from the records at our office. I wish to take this opportunity to thank Dr. Utter, Dr. Buffum and Dr. Bates for allowing me to use their case histories.

There has been a great deal written on the mode of infection in pyelitis and on the pathology of the disease. Among the most prolific of the writers has been Helmholz[1] of Rochester, Minn. He has studied the disease in the human being and experimentally in rabbits. In children he believes that there is no correlation between severity of symptoms and the pathological findings. Also that severity of symptoms cannot be used as a localizing agent as to whether there are cortical abscesses; infection of the pelvis, ureter or bladder, singly or combined.

As to the mode of infection, Beeler and Helmholz[2] found that in 50 per cent. of normal patients the urine was not sterile grown on solid media. Dr. Utter has also attempted to get a culture from the urine not infected with colon bacilli in 10 normal children, using thorough preparation of the patients with bichloride pads and so forth.

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*Read before the Providence Medical Association, November 2, 1925.

This was tried both on males and females. He succeeded in getting a sterile culture in only one of this series.

Therefore it seems that bladder cultures alone are unimportant, in that it is practically impossible to prove, even in pyelitis, that the organism comes from the kidney. Most of the large series of cases reported were all taken from the bladder alone.

Heinman[3] went a little bit further in his investigations, and in a small series of cases, 12 in number, proved by ureteral catheterization that 6 had infection of the bladder alone and of the remaining cases, 4 were bilateral infections of the pelves and 2 were unilateral.

Helmholz and Kretschner[4] concluded that ureteral catheterization didn't help much except to group cases better into upper and lower urinary tract infections. They also attempted many autopsies, but these were useless because of the rapid invasion of the colon bacillus after death into all tissues and the rapid destruction of the mucous membrane of the urinary tract. "Only on the assumption that the colon bacillus finds ideal conditions for growth in the pelvis of the kidney is it justifiable to make the assertion that it is the cause of pyelitis which no observer or research man has proven true," say Helmholz and Kretschner.

I wish to just mention by name and give the jist of the work of a few others. Bumpers and Meisser[5] produced cortical and pelvic lesions in rabbits by injecting into the blood stream Hemolytic Streptococci.

Eisendrath and Schultz[6] thought they proved that there had to be a partial obstruction and then infection ascended the ureters. Kretschner[7] thought he proved by Cystography that infection traveled by periuretral and peripelvic lymphatics.

Cabot and Crabtree[8] say that there is no question but that pyelitis can be produced by hematogenous infection.

In concluding this summary of the literature, let me quote from an article by Helmholz: "The clinical term pyelitis has been used to describe a great variety of pathological conditions all associated with pyuria. Clinically it is impossible to differentiate the different forms involving kidney cortex, pelvis, ureter, or bladder, singly or together. The pathological anatomy of these various forms of pyelitis is not well established, and does not, at the present time, allow us to determine the mode of infection except in those cases marked by cortical abscesses of the kidney. Only by careful correlation of the findings obtained by bacteriological and pathologic study as well as by experimental work can we hope to reach a better understanding of this problem."

This summary of the exhaustive labors of a few (and these were only a few) of the research men may give you an idea of how great a task we have before us in attempting to solve the puzzle of pyelitis.

I now come to the second portion of my paper, the summary of the results of the study of the 50 cases taken from private practice in this community.

Taking the cases as a whole, the age incidence varied from 3 months to 7 years; 19 cases or 38 per cent. were under one year; 16 cases or 32 per cent. were between 1 and 3 years of age; 7 cases or 14 per cent. between 5 and 7 years. It is, therefore, easy to see that the incidence grows less on advance in age. Only 3 cases or 6 per cent. were under 6 months of age, as would be expected, considering the young infant as naturally resistant to all infections. Of all these cases, only 4, or 8 per cent., were boys, but these boys had 4 of the most severe and resistant cases of pyelitis.

The most interesting phase of pyelitis, as it meets the general practitioner, is, I think, the definite clinical types which we observe. These types may present at the onset no difference, but as the disease progresses it seems to fall into one of these groups.

First: Those cases which have a sudden onset; the urine at once is loaded with pus cells; the patient becomes greatly debilitated; and the infection is very resistant to all of the well known remedies.

Second: Those cases which have as a predominant sign mucous plugs in the urine, and as an early symptom frequency of urination and dysuria. These cases seem to have more definitely an infection of the bladder than of the ureters, pelvis, or kidney tissue.

Third: Those cases having a moderate number of pus cells, singly or in clumps, in the urine and

very little frequency or dysuria. Even with the usual high temperature these children do not appear prostrated or very acutely ill. Also the signs and symptoms clear up rapidly under treatment and the infection in the urinary tract disappears in a short time.

These last are very clearly connected with upper respiratory infections. Certainly, clinically they appear to be hematogenous in origin, even though the research men have failed to recover from the pelvis of the kidney in the human being or from the kidney tissue itself in the rabbit the original infecting organism present either in the upper respiratory tract or in the blood stream. Of course, the great argument of the research men concerning this last group of cases is that the resistance of the tissues of the genito-urinary tract is so lowered that the colon bacillus finds it easy to invade these tissues and set up a pyelitis.

I believe that all cases fall fairly definitely into one of these three groups, and as the disease progresses it is very easy to classify them and keep them classified in the particular group in which they started.

I find very little in the literature on pyelitis written in the past ten years which suggests any such grouping as this. Of course, this is a very unscientific classification, but it does definitely help the clinical man in his outlook on any particular case and aid him materially in estimating what he may tell the parents in regard to duration of the disease and general prognosis.

In our series, 34 cases, or 68 per cent., fell in group 3; 14 cases, or 28 per cent., fell in group 1; 2 cases, or 4 per cent., fell in group 2.

Therefore, most of the cases fell in the group in which it seemed as if the infecting agent must be hematogenous in its origin, or at least prepare the field for the invasion of the colon bacillus.

Particular care was taken to examine the throat and nose in all of our 50 cases, and in every single patient from 3 months of age to 7 years, a definite naso-pharyngitis was found; that is, a marked redness and oedema of the anterior and posterior pillars with enlargement of the tonsils and a glistening red pharynx with enlargement of the lymph follicles. This naso-pharyngitis might or might not be accompanied by mushy oedematous adenoid tissue on digital examination and a rhinitis. There were also a few cases which had also otitis media singly or double at the time of the onset of the pyelitis. In addition, 21 cases or 42 per cent. showed definite anterior cervical adenitis, the glands varying in size from almonds to walnuts.

In 23 cases, or 46 per cent., we followed these patients in more than one attack, occasionally in as many as 10 attacks, and in every instance the train of signs and symptoms referable to the respiratory and urinary tract occurred together or within a 48 to 72 hour period.

It also might interest you to know the seasonal incidence of the disease. Nineteen cases, or 38 per cent., had their attack between January 1st and April 31st; 11 cases, or 22 per cent., between May 1st and August 31st; 20 cases, or 40 per cent., from September 1st to December 31st. This makes 30 cases, or 78 per cent., occurring in the months in which upper respiratory infections are most prevalent, although here in New England we are never free from epidemics of naso-pharyngitis at any time of the year.

Eighteen cases, or 26 per cent., gave a history of definite attacks of acute upper respiratory infection previous to their first visit at our office.

Just as an interesting observation, the temperature during the attack of pyelitis varied from 101 to 104. Thirty-five cases, or 70 per cent., reached between 104-105 during the attack.

In regard to treatment of the acute attack, we tried all the various well known methods of acidifying or alkalinizing the urine. Occasionally we were obliged to alternate the treatments changing the medium from alkaline to acid and back again. The most successful treatment was found to be that of alkalanization with a mixture of potassium citrate, drams 2 to ounces 4 of syrup of orange, giving 1 teaspoonful every 3-4 hours. We, of course, resorted to urotropin and acid sodium phosphate many times.

Tonsillectomy was done in every case over 4 years of age, and where the pyelitis had recurred more than 5 or 6 times or persisted for more than one month. There were 7 cases in this series. I would like to just enumerate the results of this procedure in the more resistant cases. Four cases were cured shortly after tonsillectomy, and have had no recurrence. In one case, a small tab of tonsillar tissue was left. Following removal of

this tab, which, by the way, was found definitely infected during each attack. After the tonsillectomy, the patient had no recurrence of pyelitis. In one case tonsillectomy was advised frequently without cooperation on the part of the parents. When finally the tonsils were removed, marked improvement resulted. There have been two attacks since that time, but the duration and severity, as shown by both symptoms and signs, has been noticeably influenced. In this case, the kidney tissue is probably so damaged that we have not yet stamped out the infection at that point. The last case has just had a tonsillectomy which was performed during an acute attack of pyelitis which had lasted over 10 days, and was the third attack to occur within a period of 6 months. The urine 48 hours after tonsillectomy was absolutely clear of pus cells, and the child was happy and had no urinary symptoms or fever.

I cannot say with too much emphasis that I believe in tonsillectomy in case of long duration or with frequent recurring attacks of pyelitis.

How do these results influence the theory that the colon bacillus ascending the genito-urinary tract is the primary cause of pyelitis and the upper respiratory infection is only secondary by its debilitating effect on general resistance. I can best reply with another question. Why does the urine become clear of pus cells so rapidly and the symptoms disappear as if by magic in such a short time following tonsillectomy?

Summary

In summarizing my report on the 50 cases of pyelitis taken from private practice in this community, I wish to state:

1. The incidence is greatest between the ages of 6 months and 3 years.

2. There are three definite clinical types of the disease:

(a) Those with sudden onset, urine loaded with pus cells, great general debility.

(b) Those with mucus plugs in the urine, frequency and dysuria.

(c) Those with mild urinary symptoms and signs, and marked upper respiratory symptoms and signs. These last being the majority of the cases seen.

3. There are definite signs of upper respiratory infection in every case of pyelitis, no matter what group it may fall in (I have omitted from this paper, of course, all surgical pyelitis where definite obstruction was found anywhere along the urinary tract).

4. The seasonal incidence, being greatest between September 1st and April 31st, points towards close association between the upper respiratory and the urinary tracts.

5. The most satisfactory treatment, in general, for all three types of cases, is alkalinization of the urine with potassium citrate.

6. Tonsillectomy is by far the most satisfactory surgical procedure in attempting to clear up the more resistant cases.

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SUGAR THRESHOLD IN ONE HUNDRED CASES OF DIABETES.

The sugar threshold in 100 cases of diabetes was determined by Joseph H. Roe and Oliver J. Irish, Washington, D. C. (*Journal A. M. A.*, May 9, 1925), and showed beginning sugar excretion levels ranging from 80 to 310 mg. of blood sugar. It is concluded by the authors that coexistence of nephritis with diabetes is apparently the cause of the very high sugar thresholds found. These facts are taken to show the importance of blood sugar findings and the insufficiency of urinary sugar examinations in diabetes.

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EDITORIALS

A PROBLEM OF MEDICAL EDUCATION.

For many years, the student in the medical school has been given a certain amount of knowledge to acquire and digest. This knowledge has been predetermined by the teacher in the medical school and by the boards of medical examiners. Are we sure that the knowledge now required of the men and women studying for the medical degree is that which they will most need when they begin the practice of their profession? Certain rather radical changes have taken place in the practice of medicine. The physician in private practice, the public health official and the institutional doctor were, in the past, called upon to treat many cases of typhoid fever. Now, typhoid fever has become almost a rarity. Tuberculosis, especially in its advanced forms, is much less frequently seen now than it was fifteen years ago, and yet, are any less hours devoted to the teachings concerning typhoid fever and advanced tuberculosis than there were formerly?

An eminent medical consultant recently stated that in 80 per cent. of the cases he saw in consultation, no physical disease could be found; another distinguished practitioner states that 80 per cent. of the cases that he sees are functional neuroses. Is it not possible that medical education should be revised somewhat along the lines of what the physician meets after he graduates from medical school rather than to have his medical program based on the traditions of the past and the special interest of the teacher? If this is done, perhaps the new doctors will have a better understanding of some of the very real needs of their patients and less about medical rarities.

A study of the case histories of a number of the physicians in our community might give us some rather striking information as to what the doctor nowadays needs to know in his practice, and it would then be very easy to determine whether the medical school had provided such information.

THE WEATHER.

To the casual reader of the future who scans the files of this JOURNAL in search of information as to the quaint ideas and crude notions of his medical ancestors, it may appear that live topics relating to the healing art must indeed have been scarce when it became necessary to ring in that subject which when introduced in drawing room conversation means the negation of intelligent thinking. To the reader of the present day, however, whose scattered calls have dragged him out night and day during the weeks just past, the subject is almost too live to be pleasant, and it must be admitted that it will take the mellowing influence of time to make some of these experiences glow in retrospect with a romance that attaches to all battles with the elements when "a human life is at stake."

The weather—worn-out topic of conersation, covering a multitude of awkward pauses—still has interest for us, the doctors. When Mrs. Brown, in explanation of little Johnny's indisposition says, "It must be the weather"—so hot, cold, damp, dry, changeable, steady or what not—we usually, as tactful inbeciles, the line of least resistance ever our guide, reply, "Oh, yes, indeed, quite so"—or words to that effect. But what, and we ask it in no flippant spirit, do we really know about the variations in the physical state of our atmospheric environment which we call weather in relation to health and disease? That an important relationship exists cannot be doubted. The premonitory twinges of the rheumatic as the glass falls and the storm approaches—a commonplace, but unexplained. The increase in the appearance of babies in wet days—a tradition, but untested. "Seasonal illnesses," so called, especially the respiratory infections, most frequent in cold weather—why?; and poliomyelitis, scourge of the summer months —for what reason? In this connection it is interesting to note the observations of the Metropolitan Life Insurance Company on 6700 cases, and more recently corroborated by the health authorities at Cornell University from a study of a large number of students, relating to the incidence of the "common cold." These investigations showed that a drop in the weekly mean temperature was accompanied by a rise in the number of colds that occurred, while with a rise in the mean temperature there was a corresponding fall in the number of colds. It is also interesting to note that the general death-rate from all causes is higher when the temperature ranges lower. It must be remembered, however, as these authors point out, that colder weather in this climate means hotter furnaces and an increased time spent in the arid atmosphere of indoors. Thus with the weather we must consider problems of ventilation and heating, problems that have been studied much and yet demand still more investigation.

THE TREND OF VETERANS' RELIEF LEGISLATION

We note with satisfaction the efforts of Senator Metcalf to have established in Rhode Island better hospital facilities for veterans. This satisfaction is born not out of state pride alone, but of the larger desire that the legitimate needs of disabled veterans shall be better served. Admitting the desirability of such a hospital being located in Rhode Island, and pledging our support to the fulfillment thereof, we must not be blinded to the dangerous trend in medical economics that "Vet-

erans' Relief" legislation is showing. Under the provisions of paragraph 10 of section 202 of the World War Veterans' Act, 1924, the federal government now treats at public expense *diseases and injuries having no relation to any government service, military or otherwise.* Regardless of the nature of their disabilities, regardless of their origin, veterans of any war, military occupation or military expedition since 1917 are eligible for treatment at government expense in government hospitals. Moreover, the government pays transportation charges incident to such treatment. The only condition the applicant must comply with is to prove his illness or injury—regardless of service origin or not—abandon his home physician or hospitals of his place of residence, and enter a government institution.

This approaches perilously communistic medicine—a political concept obnoxious to American ideals. Even the location of a veterans' hospital will not remove the evil results of this legislation in its tendency to withdraw patients from the small cities and towns and country districts to the larger cities where such government hospitals are usually located.

The medical profession yields to no other body in its ardent desire to see that every veteran receives rewards commensurate with the service rendered by him to his country during the war, but we believe that federal free medical and surgical care of veterans should be restricted to those whose disabilities have been caused by war service or to those who are unable to pay for such service.

RECENT INVESTIGATIONS OF BLOOD SUGAR*

By Philip H. Mitchell, Ph. D.

Providence, R. I.

Before the present brilliant application of the use of insulin was possible, extended physiological investigation of the internal secretion of the pancreas was necessary. Before the next step in the control of diabetes, that is its prevention and cure, can be taken, much more physiology must be known. Among the problems to be investigated is that of the mode of action of insulin. This review treats of recent work on this problem, work which seems to establish the theory that insulin causes the transformation of glucose in the body into a peculiarly useful and hitherto unrecognized form.

In order to appreciate this theory, one needs to acquire a new respect for a molecule of glucose and to learn to regard it not as a fixed, static thing, but as a peculiarly complex and changeable aggregate of atoms.

The well-known formula, long used to represent glucose, $CH_2OH{\cdot}CHOH{\cdot}CHOH{\cdot}CHOH{\cdot}CHOH{\cdot}CHO$, shows four asymmetric carbon atoms and a "free" aldehyde group; but more recent work indicates that this formula is incorrect. There are several reasons why it seems wrong: (1) If it really possessed the aldehyde group thus shown, it should be as readily oxidized as are other aldehydes. But it is not. Even under the conditions prevailing in hot Fehling's solution, considerable time is required for complete oxidization, and in the animal body glucose can circulate without necessarily being oxidized, even though certain aldehydes appear to be oxidized very rapidly. (2) Glucose shows behavior toward polarized light not explained by the classic formula; that is, it shows mutarotation (birotation). As is well known, pure glucose, freshly dissolved in water, rotates polarized light about twice as much as it does a few hours later or even a few minutes later, if warmed in a solution of the right reaction. This indicates some intra-molecular rearrangement, a tautomerism. (3) From ordinary glucose, two different varieties of forms can be separated. From a solution in 70 per cent. alcohol, there can be crystallized a form which has a specific rotation of about + 110, but which changes in watery solution to the usual form with a specific rotation of + 52.5. From hot water there can be crystallized a form with a specific rotation of about + 19, which also changes in watery solution to the usual value of 52.5. Such behavior can be explained only by assuming the presence of a fifth asymmetric carbon atom in addition to the four represented in the old formula. This and other chemical observations have led to the belief that ordinary glucose at equilibrium in watery solution is a mixture of two lactone forms known as α-d-glucose and β-d-glucose. Either of these can mutate into the other or possibly into the aldehyde

*Read before the Providence Medical Association, December 7, 1925.

form. In solution, ordinary glucose at equilibrium has about 37 per cent. of its molecules in the alpha form and about 63 per cent. in the beta form, and thus the specific rotation becomes + 52.5. If mutation into the aldehyde form is favored by alkalies, the ready oxidation of glucose in alkaline solution would be explained.

Emil Fischer and also Irvine and his co-workers have shown that there are still other modifications of glucose at least in the form of derivatives. Fischer prepared methyl glucoside, differing from the ordinary ones that can be made from the alpha and beta forms. It is of peculiar interest because it is unstable and readily subject to oxidation. Fischer called it gamma-methyl glucoside. Theoretically it should yield two forms of glucose corresponding to the alpha and beta lactone forms, but because of their instability, these two forms have not as yet been isolated. Nevertheless, they are supposed to exist and are sometimes called the gamma forms of glucose.

In 1920, Hewitt and Pryde described experiments in which they believed they had recognized gamma-glucose. They introduced solutions of pure, ordinary glucose into tied-off loops of the intestine of a living animal, and found, on removing the solutions, that they showed a rotating power too low to correspond with their reducing power. This suggested the presence of gamma-glucose. The specific rotation of the solution slowly rose, after removal from the intestine, until it came to so agree with the reducing power as to indicate the presence of the usual forms of glucose. They obtained this effect only in the living intestine. They could not obtain it in the dead one. Their work has not been satisfactorily confirmed, but it served to suggest that an especially unstable and perhaps readily oxidizable form of glucose might arise in the living body.

Two years later (1922) Winter and Smith showed that the glucose of ordinary blood had a rotating power too low for its reducing power, while the glucose of diabetic blood showed no such discrepancy. They believed, therefore, that they had detected in blood what Fischer described as gamma-glucose, just as Hewitt and Pryde believed they had detected this substance in the intestine. Winter and Smith suggested that the real failure in diabetes was the cessation of the change of relatively inert α-β-d-glucose into relatively active glucose, a change occurring in the normal body.

This work on normal and diabetic blood was repeated by several American investigators with varying success. One group of workers were quite unable to confirm Winter and Smith. It must be confessed, indeed, that the work is technically difficult. Remembering the always low concentration of glucose in blood and the necessity of removing blood proteins before analyzing for glucose, one sees difficulty in making fine distinctions; but when one notes that these distinctions involve recognition of percentages of glucose by reducing power to within about two thousandths of a per cent. and of rotating power down to the two thousandths of a degree, the exactions of the technique become apparent. All biochemists who have had experience with sugar determinations both by reducing and rotating power will agree that such exactness is not ordinarily attained. On this account the nature of the blood sugar remained in doubt.

So the matter stood for over two years, until very recently, when two Danish investigators published a series of papers reporting brilliant work which I believe opens an entirely new aspect of sugar metabolism. These writers are Lundsgaard and Holboll. They first showed that whereas glucose solutions mixed with muscle tissue or with insulin *in vitro* did not show any change from ordinary α-β-d-glucose, there *was* a change when fresh muscle tissue and insulin were both allowed to act on pure glucose solution at body temperature. The glucose, though not losing in reducing power, did lose in rotating power, so that its specific rotation fell to about one-half its usual value. On standing at room temperature, the specific rotation gradually rose until after about forty-eight hours it reached the usual value for α-β-d-glucose (52.5). If the muscle used had been dead even as short a time as two hours, it produced no effect on the glucose. The amount of the change in the glucose was increased up to a certain maximum by using more insulin and muscle substance with the glucose solution, but was more nearly proportional to the amount of muscle than of insulin; i. e., a very few units of insulin were sufficient to produce the maximum effect, but repeated

additions of considerable amounts of muscle were required for this result.

Recalling, however, that one of the common forms of glucose (the beta) has a low specific rotation (about nineteen) you naturally ask, as did these investigators, if the results obtained did not merely represent a shift of the usual equilibrium between the alpha and beta forms toward an increase of the beta at the expense of the alpha. This seemed likely, since they did not obtain any glucose with specific rotation less than nineteen.

They, therefore, undertook an elaborate investigation on a pure β-d-glucose, and proved conclusively that its behavior in coming to equilibrium (as shown by changes in specific rotation) was very different from the behavior of the form of glucose produced by the interaction of insulin and muscle. β-d-glucose under any circumstances completes its changes in a few hours, while the glucose which they discovered requires two days to complete its changes. They, therefore, proposed to call their product "new-glucose" until further investigation reveals more about its nature.

They next investigated the nature of the blood sugar in normal human blood. They were able to study this more effectively than were Winter and Smith and other previous investigators because they used a new method of determining blood sugar, that of Hagedorn and Jensen (1923). This method permits very accurate results. They found that all normal blood contained a peculiar form of glucose. In some cases, the specific rotation of the blood sugar was less than nineteen, which proved that it could not be the long known β-d-glucose. They, therefore, believe it should be called "new glucose." It could be detected in the blood under all normal conditions. Even shortly after taking 100 grams of glucose, a normal person has "new glucose" prominently present in his blood, so that a rapid change is indicated.

In diabetics, however, no "new glucose" could be detected in the blood of ten patients while they showed marked symptoms, but within two hours after administration of insulin, "new glucose" appeared in the blood.

That this transformation of glucose is essential to make it useful in metabolism is altogether likely. For instance, they showed that in every case where "new glucose" was found, the venous blood contained less glucose than the arterial, while in the absence of new glucose, the venous blood contained as much sugar as the arterial. In short, sugar was removed, presumably used, *when present as "new glucose"*—but not otherwise.

"New glucose" was also found in spinal, edoema and pleural fluids from persons showing normal sugar metabolism.

What is "new glucose"? It may be gamma-glucose, which Fischer postulated to exist, but which has not been proved to occur except as glucosides. This does not seem very likely, since "new glucose" outside the body reverts to ordinary α-β-d-glucose in about two days, and without decrease in amount. Such behavior would not be predicted for the very unstable gamma forms. "New glucose" may be nothing else than the simple, free-aldehyde form which we used to suppose was that of ordinary glucose. This point will require investigation.

Indeed, one is struck by the many new lines of investigation opened up by the discovery of "new glucose." Where in the body is it formed? Do the intestinal wall and the liver react with the aid of insulin to form new glucose as well as do the muscles? Is new glucose better able to penetrate living cells than is ordinary glucose? What is the nature of the substance contributed by muscle to aid new glucose production? Is it perhaps some unstable protein which changes after the death of the cell? How is glycogen related to new glucose production? Must carbohydrate food go through the glycogen stage before conversion to new glucose?

These and many other problems await investigation. When they are solved, new light will be thrown upon carbohydrate metabolism, and so, indirectly, upon the nature of diabetes.

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HOSPITALIZATION OF WAR VETERANS TODAY

By Inez M. Pugh

Notwithstanding all that has been said and written about hospitalization of disabled American War Veterans, how many individuals in the United States today have any definite idea of the elaborate hospital program that the U. S. Veterans Bureau is carrying on?

Already operating 49 hospitals, 74 dispensaries, 94 clinical laboratories, about 100 X-ray laboratories and housing over 29,000 patients, the Bureau is constantly constructing and opening new hospitals and incorporating additional facilities in those already open. These hospitals are as modern and complete as science and careful planning can make them and no detail of utility or convenience is sacrificed to a false prompting toward economy.

In order that the medical authorities of the hospitals may be enabled to give their undivided attention to the care and treatment of patients the Director has established a business manager in each hospital to look after the financial and economic affairs of the institution.

These men have been carefully selected with regard to demonstrated ability as business executives and are expected to show gratifying results in the way of increased economy of administration, and in handling the thousand and one business details inseparable from the functioning of a large institution.

It has long been the Director's conviction that these duties should not be imposed upon the medical men charged with the actual care and treatment of the disabled and the establishment of these business managers is a definite gesture planned to increase efficiency on the part of the physicians as well as in the economic operation of the hospital.

In the matter of hospitalization of disabled veterans the President, the Director of the Bureau and the Congress are thoroughly in accord and whatever may be necessary in material and personnel to furnish adequate hospitalization and medical service of the highest order is being and will be provided for. The generous provisions of the Reed-Johnson Bill have permitted the Bureau to open its hospitals to veterans of any war in which the United States has participated since 1897 and already over 2,000 have availed themselves of this benefit showing plainly the acute need for such assistance.

In planning the hospitals, not alone is the medical care of the men considered, but recreational and entertainment features are also provided, chief among which latter are the radios which are being installed in all Veterans' Hospitals as rapidly as suitable equipment can be obtained.

In the appropriations recently made available by Congress complementing the Third Langley Bill, six new hospitals and a National Training School for the Blind are provided for and funds are made available for the completion of another hospital now partially constructed.

In order to secure for the Bureau the greatest possible efficiency in medical service the Director has assembled a body known as the Medical Council which is composed of thirty of the leading specialists of the United States and which meets at his call to counsel and advise with him and the Medical Director in all matters pertaining to the medical care and treatment of the disabled.

The Director feels that it is much more a service to give a man back his health and with it his economic independence than it is merely to maintain him in a hospital and pay him compensation. Therefore, this feature is a significant step in demonstrating his theory that cure rather than money compensation should be the chief endeavor of the Bureau.

In this theory the Medical Council heartily concurs and in accordance with this policy a hospital's efficiency is measured by its accomplishment in recoveries of the disabled.

In this phase of the work, however, the attitude and co-operation of the patients is half the battle and if past experience is a safe criterion for the future the outlook is indeed encouraging.

The boys who had the courage and grit to carry on throughout the war are demonstrating that same spirit in their slow and irksome fight back to health and strength and in each recovery credit for the victory belongs quite as much to the patient as to the physicians and nurses.

In many of the Bureau hospitals the men find much pleasure, healthful exercise and recreation in the planting and tending of truck and flower gardens. This occupation is always encouraged and provisions for various other forms of occupa-

tional therapy are constantly being developed in the hospitals.

In a great many of the hospitals, a small weekly or monthly paper is edited and published entirely by the patients and personnel and many of these papers show genuine merit in carefully prepared articles which are a faithful reflection of the fine spirit prevailing in the hospitals, as well as many amusing little local squibs which record the daily life at these great institutions.

There was an old fashioned idea that a hospital was a gloomy, disinfected place, redolent of iodoform and hung with fever charts, in which to be sick and do something about it, but this notion has given place to a gratifying knowledge that the Veterans' Bureau Hospitals at least, are "comfy," cheerful and pleasant, and that mental contentment for the patients is quite as important an objective as physical relief and betterment.

There is a certain personal quality in the service that the physicians and nurses render the disabled as though they bear constantly in mind with grateful remembrance the cause and source of the wounds and hurts they strive to heal.

It is on such a basis as this that there has been built up in the U. S. Veterans' Bureau Hospitals a morale and an esprit de corps of which both the patients and the personnel are justly proud, and upon which most surely rests the success of these institutions.

U. S. Veterans' Bureau

SOCIETIES

Providence Medical Association

The regular monthly meeting of the Providence Medical Association was called to order by the President, Dr. Roland Hammond, Monday evening, March 1, 1926, at 8:50 o'clock. The records of the last meeting were read and approved.

A letter from the Providence Committee, American Foundation for the Blind, was read, urging attendance at a talk by Miss Helen Keller.

Dr. Van Benschoten reported a case of a boy pecked in the eye by a rooster, pulling part of the iris to the surface. Operation resulted in a good eye.

Dr. Mowry reported that the walls of the dining room had been refinished and cautioned the members against marring them.

Dr. Frederick N. Brown gave helpful advice.

The first paper of the evening was on "The Care of Diabetics at the R. I. Hospital Since 1910," by Alex. M. Burgess, M.D.; Louis I. Kramer, M.D.; Miss Miriam J. Carpenter, Miss Helen S. Munroe. Dr. Burgess read the paper.

The two great advances in this period have been the systematic instruction of patients and the use of insulin. While the incidence of the disease seems increasing, the span of life is increasing also.

The period under discussion he divided into three parts: when treatment was as before; the period of under nutrition with instruction, and the period with insulin. In the first the light cases could be improved, but were always back sliding; in the second the severe cases were held in check, but dragged out a miserable existence; and in the third the severest cases lead a practically normal life. Hospitalization is now a mere incidence, with a large out patient clinic. Before insulin, no coma cases recovered. Now, with the use of very large doses of insulin, recovery can be assured unless there are complications. Surgery now can be done as indicated irrespective of diabetes. In conclusion, he outlined detailed routine improvements in technique, plans for which are now being matured. Dr. Louis I. Kramer stressed particularly the care of coma and reported cases.

The discussion was continued by Drs. Wells, Mathews, Streker, Mowry, Gerber, Burgess, Van Benschoten and Leonard.

The second paper was on arterio-sclerosis by Dr. Clinton S. Westcott.

This is best described as a thickening of the arterial coats with degeneration diffuse or circumscribed. It is an anatomical rather than clinical entity. Its forms differ in general according to the size of vessels affected.

The causes seem to be the wear and tear of life and intoxications. Diagnosis is of a double nature, as to its presence and activity.

The signs and symptoms are largely dependent on the disfunction of organs due to impaired blood supply; in active cases, hemorrhage and pain are the characteristics.

Treatment resolves itself into keeping bodily activities at a low pain. The iodides, nitrites and particularly sedatives are of value.

To secure results we must anticipate.

The paper was discussed by Drs. Mathews, Gray, Mowry and Westcott.

Meeting adjourned at 10:15 P. M.

Attendance, 71.

Collation was served.

Respectfully submitted

PETER PINEO CHASE
Secretary

RHODE ISLAND MEDICAL SOCIETY

The regular quarterly meeting of the Rhode Island Medical Society was held Dec. 3, 1925, at 4 P. M. at the Medical Library, Providence, the President, Dr. DeWolf, presiding.

The minutes of the September meeting of the Rhode Island Medical Society, and the minutes of the November meeting of the Council and House of Delegates were read by the secretary, and approved.

Dr. Richardson reported on the Clinical Conferences.

Dr. DeWolf announced that all the members were invited to a meeting Dec. 10 at 8 P. M., at the Medical Library, of the Rhode Island Ophthalmological and Otological Society. A paper to be read by Dr. Harry P. Cahill, Chief of Aural Service, Boston City Hospital, on "Brain Abscess Complicating Aural Disease."

Dr. J. W. Keefe spoke about having the Library properly catalogued to the advantage of all. A motion was made by Dr. Leech, and duly seconded, that this matter be referred to the Committee on Library. So voted.

Dr. DeWolf expressed his pleasure at seeing so many members from outside of Providence present at the meeting, and hoped for a continuance in larger numbers.

The following papers were read:

1."Measles Immunization," Dr. H. P. B. Jordan; discussed by Dr. D. L. Richardson, Dr. W. P. Buffum, Jr.

2. "Diagnosis and Treatment of Gall Bladder Disease," Dr. J. B. Ferguson; discussed by Dr. J. F. Boyd with slides, Dr. C. O. Cooke.

3. "Deep X-Ray Treatment — Its Development and Present Status," Drs. I. Gerber and S. Albert; discussed by Dr. H. C. Pitts.

4. "The Infected Kidney — Its Physiology, Pathology and Treatment," Dr. Clyde Leroy Deming, Clinical Professor of Surgery, Yale School of Medicine; discussed by Dr. J. E. Kerney, Dr. E. Stone, Dr. J. A. McCann.

After adjournment a collation was served.

J. W. LEECH, *Secretary*

The regular quarterly meeting of the Rhode Island Medical Society was held March 4, 1926, at 4 P. M. at the Medical Library, the First Vice President, Dr. H. G. Partridge, presiding in the absence of the President.

The minutes of the December meeting and of the two special meetings of the House of Delegates of Jan. 12th and 13th were read by the secretary pro tem., Dr. Peter P. Chase, who was elected in the absence of the secretary on account of illness, and approved.

The First Vice President called attention to the coming meeting of the American Medical Association at Dallas, Texas, in April, and directed the attention of the members to announcements of rates and accommodations by several of the railroad companies posted in the reading room of the Library.

The first paper was on "The Spinal Fluid," by Dr. John E. Donley, Providence.

The second paper was on "Blood Transfusion," by Dr. William P. Davis, Providence; discussed by Drs. McDonald and Cameron.

The third paper was on "Treatment of Suppurative Conditions of the Lung," by Dr. Wyman Whittemore, Assistant Surgeon, Massachusetts General Hospital, Boston; discussed by Drs. Matteson, Kelley, and Gerber.

The presiding officer stated that a bill to regulate the practice of medicine, and known as the Healing Art Bill, had been presented to the House of Delegates at its meeting on Jan. 12th and had received the approval of the House of Delegates; that since the introduction of this bill into the legislature so many changes had been made in the original bill as in the opinion of the Committee on Legislation to vitally change the purpose and in-

tent of the bill from that which the House of Delegates had approved; and that the Committee on Legislation did not feel justified in extending to the altered bill, known as the Clifford Substitute A Bill, the support which the House of Delegates had voted to the original bill. It seemed desirable that further consideration of the bill as now in the legislature be given it by the House of Delegates. Dr. Bugbee moved that a vote of confidence of the Society be given the House of Delegates in its action in regard to pending legislation. After discussion by Drs. Ventrone, Brown, Kelley, Mowry, Hawkins and Skelton, the motion was lost. On motion of Dr. Skelton, duly seconded, it was voted that a committee of five be appointed to consider a memorial to Dr. Chas. V. Chapin. A rising vote of thanks was attended Dr. Whittemore for his kindness in appearing before this Society.

After adjournment a collation was served.

P. P. CHASE, M.D.,
Secretary pro tem.

HOUSE OF DELEGATES

A special meeting of the House of Delegates was called by the First Vice President, Dr. H. G. Partridge, in the absence of the President, at the Medical Library, at 11 o'clock A. M., March 5, 1926. Dr. P. P. Chase was elected secretary pro tem. in the absence of the secretary on account of illness.

The purpose of the meeting was to review and take action upon the so-called Healing Art Bill, S-51 Sub. A. Dr. Fulton explained that this bill is the substitute which the Senate Committee on Judiciary has introduced in the place of the Healing Art Bill which the House of Delegates approved at its meeting on Jan. 12th, 1926. He pointed out the many and important changes which had been made in the original bill, whereby the intent and purpose of the bill as it appeared to the House of Delegates at its meeting on Jan. 12th had been largely nullified. He stated that it is no longer the same bill in intent or purpose and that it did no longer meet with the approval of the Committee on Legislation. He, therefore, moved and it was duly seconded that the House of Delegates disapprove of bill S-51 Sub. A, the so-called Healing Art Bill now before the state legislature. So voted.

Adjourned.

Dr. PETER P. CHASE, *Secretary pro tem.*

RHODE ISLAND SOCIETY FOR NEUROLOGY AND PSYCHIATRY

The February meeting of the Rhode Island Society for Neurology and Psychiatry was held at the home of Dr. George L. Shattuck, 150 George Street, Providence, R. I., on Monday evening, February 8, 1926, at 8:30 o'clock.

Dr. Harrington made a brief report of the bill before the legislature for the establishment of a hospital for the criminal insane.

Dr. Paul J. Ewerhardt was elected to membership. The program was as follows:

"The Role of the Vegetative Nervous System in Determining Some Organic Nervous Diseases," Dr. Harvey B. Sanborn. This paper was discussed by Dr. Charles A. McDonald and Dr. John E. Donley.

Report on the New York meeting of the Association for Research in Nervous and Mental Diseases, by Drs. Charles A. McDonald, William N. Hughes and Harvey B. Sanborn. Following these reports, informal discussion took place.

Following the meeting refreshments were served by Dr. Shattuck and on motion of the President, Dr. Harrington, a vote of thanks was extended to Dr. Shattuck for his hospitality.

HOSPITALS

The following is a report of the March meeting of the Memorial Hospital Staff.

"Meeting held March 4, 1926.

"Meeting called to order at 9:00 P. M. by President Wheaton. Minutes of the January meeting were read and approved. Members present: Drs. Wheaton, Shaw, Jones, Kerney, McGraw, Marshall, Boyd, Bates, Triedman, Saklad, McLaughlin, Lutz, Moor, Miller, Holt, Towle, Sprague and Kenney. An interesting paper on 'Gall Bladder Examination' was read by Dr.

James F. Boyd. Remarks on record system made by Dr. A. Miller. Motion was made and duly seconded that the President appoint a Committee to consider ways and means to keep up records and to suggest rules, etc., to Trustees. Committee: Dr. A. T. Jones, Chairman; Dr. A. Miller, Dr. J. L. Wheaton. Meeting adjourned at 10:10 P. M.

"JOHN F. KENNEY, M.D."

MISCELLANEOUS

THE USE OF PHYSOSTIGMIN IN ABDOMINAL DISTENTION.

The use of physostigmin in abdominal distention was studied by Hayes E. Martin and Soma Weiss, New York (*Journal A. M. A.*, May 9, 1925), in nontoxic cases in which abdominal distention followed laparotomy, surgical shock, early intestinal obstruction, or injury to the central nervous system, and in toxic cases, in which the condition was associated with peritonitis or general toxemias, such as pneumonia or long-standing intestinal obstruction. In every case various simple measures, such as gastric lavage, enemas, turpentine stupes and colonic irrigation were employed when distention occurred, and physostigmin was not used unless those measures prove ineffective, the purpose being to learn whether physostigmin is capable of relieving these patients. All the patients who received physostigmin were in a serious conditions, and all manifested alarming symptoms. All of the sixteen patients embraced in the group of nontoxic cases of abdominal distention were completely relieved of this distressing symptom by the injection of physostigmin. The fifteen patients embraced in the toxic group were benefited but little or not at all. The results of these experiences indicate that the drug should be administered in doses sufficient to induce its characteristic therapeutic effect or until the occurrence of systemic actions indicates that the limits of safe dosage have been reached. Physostigmin is a useful drug for the treatment of abdominal distention in nontoxic cases, especially. It is less useful in cases of the toxic type. It has fallen into disuse mainly because of the employment of insufficient doses, partly, perhaps, because of the failure to distinguish the type of cases in which it is more effective (nontoxic type) from those in which it is less effective (toxic type). The effective dose of the salicylate or benzoate, in cases which do not yield to simple measures, is from 3 to 4 mg. (from 1-20 to 1-16 grain) injected intramuscularly. Such a dose may be repeated once after an hour if the first does not induce any systemic effects, and it may be repeated three times (at least) at intervals of three or four hours if there are no symptoms which indicate that its systemic effects persist. The general condition of the patient and his behavior toward the drug must be observed carefully and must serve as a guide for the repetition of the dose. The dose required for those patients who respond to simple therapeutic measures, such as rectal enemas, has not been determined.

ANTAGONISTIC ACTION OF POSTERIOR PITUITARY EXTRACT AND INSULIN.

From work performed on diabetic patients, Robert C. Moehlig and Harriet B. Ainslee, Detroit (*Journal A. M. A.*, May 9, 1925), believe that pituitary extract injections improve the muscular asthenia to a great extent. This is true despite the fact that the patients, for the purpose of the work, are not placed on a diet. Patients with hypopituitarism suffer from asthenia, and fatigue is easily induced. The opposite is true in cases of hyperpituitarism. Posterior pituitary extract injected into normal rabbits produces, as a rule, a slight rise in blood sugar. Posterior pituitary extract, when injected simultaneously with insulin, prevents the fall produced by the latter. Posterior pituitary extract, injected during insulin hypoglycemic convulsions, produces a rapid rise in blood sugar, with subsequent recovery of the rabbits. The point of attack of the pituitary extract seems to be in the periphery; viz., the skeletal muscle metabolism.

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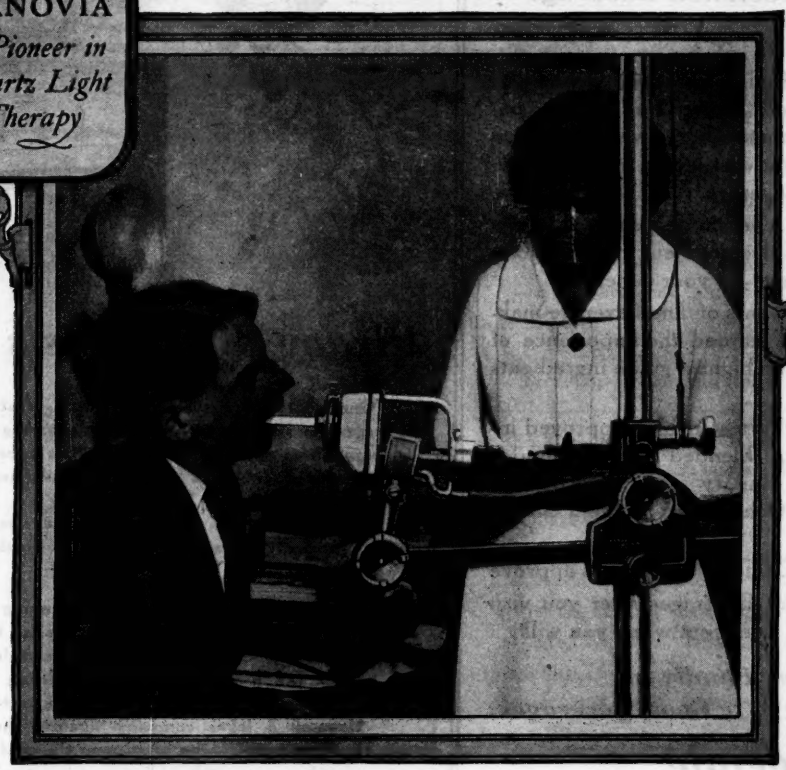

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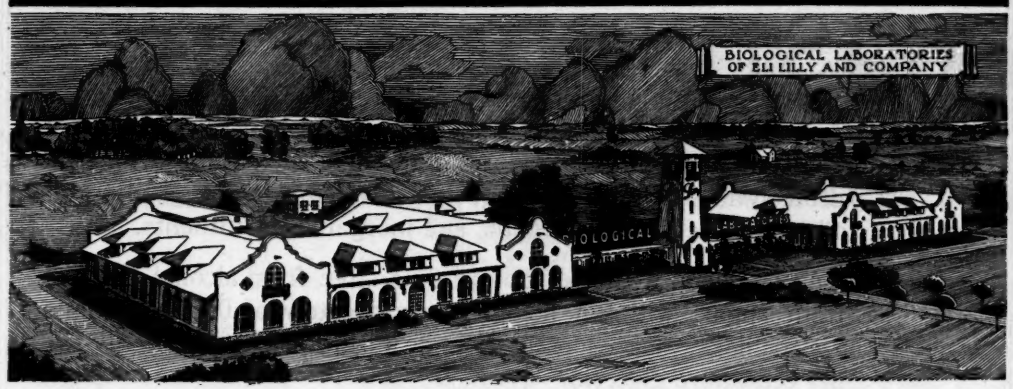

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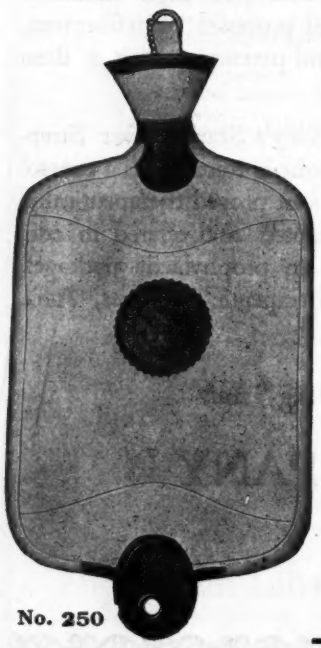

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